The frequency of accessory portions of thyroid tissue has been well known since the work of E. Zuckerkandl,[1] but that they must be much more frequent than even he thought is well shown by the facility with which they were found in the specimens we have studied. Thus of the two hundred and sixty-three specimens one hundred and seventeen were found to contain thyroid tissue in large part, most of these being sections of small accessory nodules, and these it should be remembered included only those nodules selected because of a resemblance to parathyroids; there were many others.

Remnants of the thymus gland were also found to be present in considerable number, and occurred in the aged as well as the young, though they were of course present in largest number in infants, as shown in the above table.

FOUR CASES OF DOUBLE HEMIPLEGIA (SYPHILITIC ENCEPHALITIS, CEREBROSPINAL SYPHILIS), WITH AUTOPSY.[2]

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THE following four cases of double hemiplegia illustrate some of the pathological changes which may occur in the nervous system as a result of syphilis. The pathological conditions commonly met with in cerebral syphilis are: gummatous formation; meningitis; disease of the bloodvessels; and areas of softening, usually the result of vascular disease. Less frequently syphilitic encephalitis occurs, although this is undoubtedly more common than some believe.

The first case (from the service of Dr. Wiliam G. Spiller, at the Philadelphia General Hospital) illustrates several of these processes, causing an infrequent train of symptoms, and is further interesting because an exact pathological diagnosis would have been impossible without a careful microscopic study of the specimens.

CASE I.—A woman aged fifty-four years, a domestic by occupation, was admitted to the Philadelphia General Hospital, April 2, 1901, suffering from right-sided hemiplegia.

Her father died of old age and her mother of phthisis. She had had scarlatina and pertussis when a child, and in the winter of 1901 she had an attack of pneumonia. Three months prior to her admission she was suddenly seized with unconsciousness, lasting ten minutes. No paralysis followed this attack, but a few weeks later, upon arising in the morning, she complained of dimness of vision and difficulty in expressing herself. A week later, and a month before she was admitted to the hospital, the right arm became weak and numb, and the right leg was slightly weaker than the left. There was no palsy, but the tongue protruded to the right.

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[1] Stuttgart, 1879.

[2] Read before the Philadelphia Neurological Society, April 5, 1905.

When examined a week after her admission there was decided weakness of the whole right side—arm, hand, and leg—and she dragged her right foot when walking. The tongue was protruded to the right, and she had some difficulty in expressing herself. She used the wrong words, and miscalled the names of objects, though after making several attempts she would finally recall the proper word. She would call a pocket-knife a "screw-knife," and a pen a "pencil to ink with." There was slight hyperæsthesia on the right side below the knee, and sensation was blunted in the right arm. The right arm and leg were both spastic.

All the reflexes were exaggerated. The knee-jerks and the biceps-jerks on both sides were increased, but more so on the right than on the left side. The Babinski phenomenon could not be obtained on either side at this time. Her lungs and abdomen showed no abnormalities. The pulse was slow and rather weak, but regular.

On April 14 she fell twice, without apparent cause, and without disturbance of consciousness. After the last fall the left arm was found to be weak and spastic. The pupils were equal and responded to light, and there was no hæmianopsia. There was no sensory change. The Babinski reflex was present on both sides. Three days later the grasp was nil on the left, and she lifted her arm with difficulty. The knee-jerks were increased on each side, but more on the right. The left leg was at this time weaker than the right. There was no abnormality of the heart and lungs.

On April 27, ten days later, she fell again, and upon assisting her to her feet it was found that there was some loss of power in the legs, that she could not articulate distinctly, and that the face was flushed. The right leg was spastic and the knee-jerks were increased. The Babinski reflex was present, but there was no clonus.

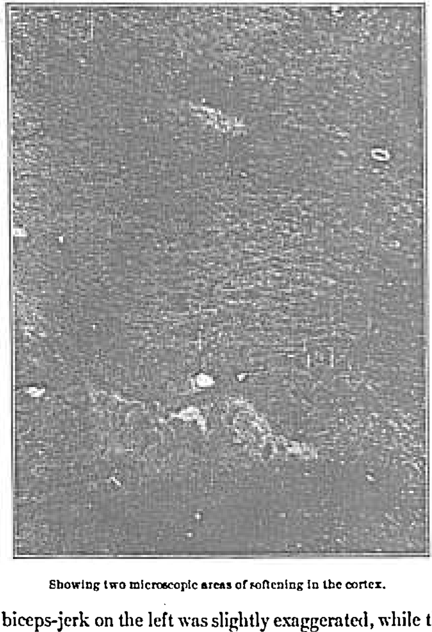
She was examined by Dr. William G. Spiller, July 6th, and the following conditions were found: Both feet were contractured in the position of talipes equinovarus. The contractures, however, could be overcome by passive movements, but were more marked on the right side than on the left. When the legs were elevated and allowed to drop they fell as if paralyzed. In the few days preceding the examination there had been incontinence of urine. There was no marked atrophy.

The knee-jerk on the right side was prompt, but not exaggerated, and on the left it was absent, and no movement of the quadriceps muscle could be seen. The Achilles jerk on both sides was absent. Babinski's reflex was present on both sides to a marked degree.

When stuck with a pin the patient made no response, excepting when the sole of the foot was tested.

The right arm was in the position of an old hemiplegia. The fingers were slightly flexed, especially toward the ulnar side. The hand was extended at the wrist, and the forearm was flexed to a right angle on the arm. This contracture could be overcome by passive movements.

FIG. 1



Showing two microscopic areas of softening in the cortex.

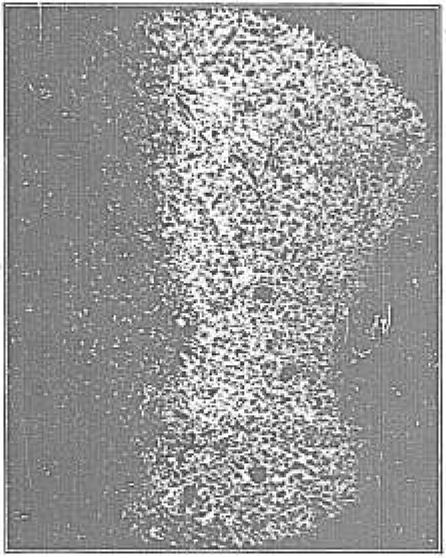
The biceps-jerk on the left was slightly exaggerated, while the olecranon and wrist-jerks could not be obtained. The arm was spastic, and when allowed to fall showed increased tonicity.

The face and head were drawn to the right. There was marked conjugate deviation of the eyeballs. The pupils were equal and the eyelids could be closed. When the lid of the right eye was irritated the eyelid closed firmly. When the left lid was irritated the lid was closed less firmly than on the right side. There was no puffing of either cheek indicative of facial palsy. The patient died of pneumonia.

The pathological diagnosis was lobar pneumonia; chronic nephritis; cirrhosis of the liver, and arterial sclerosis.

The brain and spinal cord were placed in Müller's fluid, and sections from various portions were stained by the Weigert hæmatoxylin method, and by hæmalum and acid fuchsin.

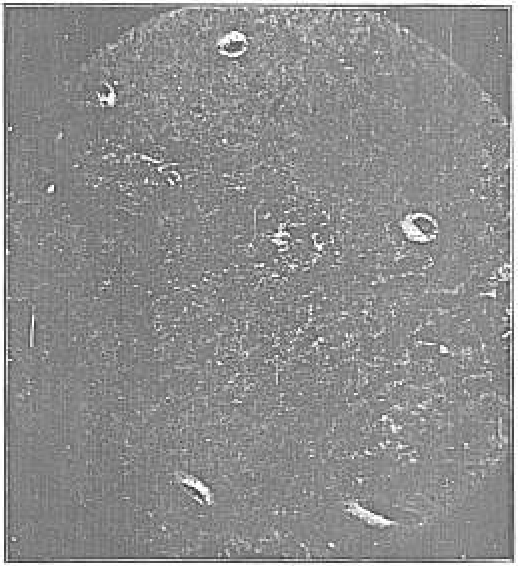
FIG. 2



Area of softening in the cortex showing the fatty granular cells. (Higher magnifications of areas shown in Fig. 1.)

In the right paracentral region there was a pronounced meningitis. The cells of Betz were distinctly diseased, the bodies of the cells being swollen and the nucleus displaced. In the cortex, just below the surface, were seen several small areas of softening, consisting of masses of fatty granular cells, closely packed together.' (See Figs. 1 and 2.) In places these fatty, granular cells extended within the tissue, along the outer layer of the cortex just below the surface, without forming any distinct area of softening. Elsewhere in the cortex in the paracentral region there existed much round-cell infiltration, around the vessels especially, and elsewhere more moderately. The perivascular spaces were distended, and everywhere in this region the bloodvessels were markedly congested. A few small hemorrhages were present. In the right occipital region a round-cell infiltration of the pia was observed. No conspicuous change was noted in the cortex.

FIG. 3



Area of inflammatory encephalitis; distribution of perivascular spaces; round-cell infiltration; congested vessels.

In the right frontal region the pia was intensely infiltrated. The cells consisted mainly of mononuclear round cells, but a few large cells were found containing two or three nuclei. The pia was much thickened, and there were, in places, newly formed bloodvessels. The vessels of the pia showed distinct change. In some of these the disease was more intense in the adventitia, and the intima, while showing some proliferation, was less affected. In others all the coats of the vessels were thickened, and the proliferation of the intima was so intense that the lumen was almost obliterated. One large vessel, the walls of which were thickened, was obliterated entirely, save for four small lumens around which the exudate was more deeply stained, suggesting the formation of denser tissue in these places. In the cortex, just below the surface corresponding to the site of an intense meningeal process, the nerve tissue was loose, and the normal appearance could not be made out. Small holes were seen here and there, probably due to resorption of the fibres, or the result of œdema. A little deeper, in the white matter, a focus of inflammation was observed. Here there was intense, round-cell infiltration, and the vessels were much congested. This area was more or less circumscribed, and the tissue around it was undamaged. (See Fig. 3.)

Elsewhere in the cortex distention of the perivascular spaces was present, and the vessels were generally congested. An intense meningitis was present around the optic commissure, and the bloodvessels within the commissures were much congested. Here, also, were found enlarged perivascular spaces and round-cell infiltration. No degeneration of the fibres of the optic commissure was discovered. No degenerated fibres could be seen in the pyramidal tracts of the pons, stained by the Weigert method, but when stained by the Marchi method quite a considerable degree of acute degeneration was seen on one side.

The pia over the cerebral peduncles was the seat of an intense, round-cell infiltration, especially marked around the vessels. A similar condition was also found in the pia of the medulla oblongata. By the Weigert stain no degeneration of the peduncles was observed. In the pons there was an area of softening $\frac{1}{8}$ x $\frac{1}{4}$ inch in area, just above the median fillet.

In the medulla oblongata the left anterior pyramid took the Weigert stain a little less deeply than the right. Considerable acute degeneration was seen, however, in the sections stained by the Marchi method. The right crossed pyramidal tract and the left direct tract in the cord showed, by the Weigert method, a slight degeneration.

Summary of the Pathological Findings. There was present a round-cell infiltration of the pia, with disease of the bloodvessel walls, especially marked in the frontal region, but present at the base and convexity as well. In the paracentral cortex small areas of softening were present, and in the frontal region there were areas of inflammatory encephalitis. The cortical vessels were congested, and round-cell infiltration and distention of the perivascular spaces were common. The cells of Betz were diseased. In the pons and the medulla oblongata recent degeneration of the nerve fibres was seen, and in the medulla oblongata degenerated fibres were discovered by the Weigert method.

The bloodvessel change calls for some discussion, as the findings were not entirely characteristic of the description of syphilitic arteritis as laid down by various authorities. Some of the vessels showed a preponderance of the disease in the adventitia and media, while in others all the coats of the vessels were greatly involved, the lumen of the vessel being frequently almost entirely or quite obliterated by the proliferation. In some parts of the brain, the occipital region principally, the vessels showed little change. But wherever the meningeal process was intense the bloodvessels were much diseased.

Heubner[13] claims that syphilitic disease of the vessels originates between the endothelium and the membrana fenestrata. Round cells escape later from the vasa nutritia of the vessels, giving rise to the granulation tissue of the intima. According to Friedländer,[10] the process is an endarteritis obliterans. On the contrary, Baumgarten[4] believes that the primary alteration arises in the adventitia and its vascular apparatus, causing secondary thickening of the intima. Baumgarten[5] has described disease of the bloodvessels in syphilis, consisting of gummatous deposits between the adventitia and the media, and in both of these coats of the vessel resembling in structure a meningeal node. He claims that occasionally the deposit within the vessel directly communicates with these nodes. He found instances in which there existed periarteritis and mesarteritis without proliferation of the endothelium. Oppenheim[28] reported a case also in which the disease of the vessel originated directly from a gummatous tumor. On the other hand, the vessels may alone be diseased, as Marchand and others have described. Rumpf looks upon the change in the intima as a reactive proliferation, having its origin in the vasavasorum. Köster[16] also is of the opinion that the origin of the process is in the vasavasorum of the adventitia, and that the inflammation of the muscularis follows with the escape of lymphoid cells, giving rise to a mesarteritis, and, finally, an endarteritis. Oppenheim[27] believes that in the majority of cases the specific meningitis attacks the vessels directly, and may involve one or all of the coats.

A study of the change in the vessels found in my case sheds little light upon this mooted question, though the evidence may point to the conclusion that the process sometimes involves primarily the outer coats, when it would seem to be a part of the pathological change in the meninges, or that the intima may be primarily attacked, the inflammation arising from direct irritation of the poison circulating in the blood.

The cases of syphilitic encephalitis in the literature, with a few exceptions, are so lacking in the description of the finer histological changes that satisfactory deductions cannot be made.

Cases reported as syphilitic encephalitis are often merely examples of cerebral softening; others lack microscopic description, and are therefore valueless; while still a very few other references are found to what may be termed a true inflammatory encephalitis.

As illustrating the first group of cases, the case of Obermeier may be cited. In his case softening of the cortex was present beneath gummatous inflammation of the meninges. Griesinger[11] collected a number of cases in which foci of softening were present in the brain, probably of vascular origin. He stated that the apoplectic form of brain disease in syphilis was in the main due to encephalitic foci, and that hemiplegia was often the result.

Other cases have been recorded by Tournier,[31] Mildner,[22] von Bechterew,[3] Lancereaux,[18] and Rosenthal,[30] while Nonne,[24] Ljunggren,[19] Gowers,[12] and Naunyn[25] mention softening in the brain as a frequent occurrence in brain syphilis. According to Oppenheim,[27] in the majority of cases the brain softening originated in obliterated vessels, or in consequence of a thrombosis. Again, softening has occurred around gummas, while specific meningitis has given rise in some cases to the same condition.

I could find no case except that of Barrett[2] in which the post-mortem findings were comparable to the small areas of softening in the cortex in my case, most of the cases reported being examples of extensive foci of softening, readily seen by the naked eye. The cortical areas of softening I have described are remarkable in that they are detectable only with the microscope. But in most of the cases of softening reported microscopic descriptions are lacking.

Encephalitis of inflammatory origin in syphilis is a less common, but by no means a rare, occurrence. Gowers[12] regarded inflammation of the brain of specific origin, however, as rare, and while admitting that primary softening and induration had been met with, there was always the question, are they not of vascular origin. They are, he says, seldom specific when inflammatory. In Barrett's case of syphilitic encephalitis the findings were in many respects similar to those found in my case. There were present, in his case, an inflammation of the pia-arachnoid, and proliferation and degeneration of the vessels. In the vessels the intima was most frequently affected, while the adventitia was more involved than the media. The nerve cells were degenerated, and proliferation and degeneration of the glia and vessels of the cortex were present. There were also areas of encephalitis, composed of granulation tissue, besides foci of simple necrosis related to a neighboring vessel. Besides these a few gummatous foci and some hemorrhages were found.

Oppenheim[28] reported a case of softening independent of blood-vessel change. Two foci were observed in the brain, showing the presence of round-cell infiltration, plasma cells, "körnchenzellen," destruction of nerve tissue, and new vessel formation. He compared this to the encephalitis of Hayem and Friedmann. In a case reported by Charcot and Goumbalt[7] two foci were seen in the medulla oblongata, containing a number of cells which appeared to be highly developed neuroglia cells. There seemed to be a proliferation of the neuroglia cells, which, pressing against each other, ceased to be properly nourished, and degenerated in consequence.

In Obermeier's[20] case there was a proliferative and degenerative process in the pia, and in the cortex the pia cells besides being changed in form were degenerated. The neuroglia was proliferated, and there was a round-cell accumulation. Elsewhere the perivascular spaces were distended, and areas of circumscribed infiltration were seen. The bloodvessels were intensely diseased, and some of them were obliterated.

Alzheimer[1] reported a case in which the cortex was the seat of a high-grade infiltration. The vessels were narrowed or obliterated; perivascular infiltration was present, and the nerve cells were altered. There was, in a case of Möller,[21] inflammatory infiltration of the dura, and besides thrombosis of the artery of Sylvius there was found "sclerosis" and degenerative change in the basal ganglia.

Cases of encephalitis (so reported), with less satisfactory descriptions, are those of Tugendreich,[23] in a child one month old, Schaffer,[22] Jastrowitz,[14] and Köbner.[17] Heubner[13] collected from the literature up to 1877, one hundred and fifty cases of simple inflammation of the brain of syphilitic origin, of which he found only twenty-four trustworthy cases.

In connection with the study of the foregoing case of double hemiplegia, an opportunity to study three other cases, in the Philadelphia General Hospital, was offered by the courtesy of Dr. William G. Spillier.

Case II.—G. S., aged sixty-nine years. Family history negative. He denied specific infection, and gave a history of vertigo for two years previous to admission. On the day before admission (September 16, 1904) he had a dizzy attack and fell, although he was not unconscious. Dizziness and headache followed. Since that time he has not been able to walk. When examined later (the notes do not say exactly when) the left side of the body was spastic, especially the leg; the reflexes were generally exaggerated, although they were not distinctly elicited on the left side on account of the spasticity. Babinski's sign was observed on both sides. He urinated involuntarily, and complained of shooting pains. There was also some tenderness over the nerve trunks. Three months later (December 22, 1904) after "feeling badly" he found he was unable to feed himself with the right hand. Distinct weakness of the right arm and leg was noticed, associated with some difficulty in articulation. The speech was rather explosive, although no true aphasia was demonstrated. The grasp on the right side was weak, and the muscular power of the arm was lowered. The reflexes were prompt in the right arm, while in the right leg the knee-jerk was exaggerated. No patellar or ankle clonus could be elicited. Babinski's sign was prompt on both sides.

There was no anæsthesia or paræsthesia present. The tongue was protruded to the left, and the pupils were normal and responded to light and in accommodation.

When examined on March 22, 1905, I found the patient in the following condition: He was bedfast, and unable to move the arms or legs. When spoken to he made no sign that he heard, nor did he obey commands. He would not protrude his tongue, and made no effort to move his arms or legs, or to wrinkle his brow. However, he chewed his food, and the act of deglutition appeared to be unaffected. The arms and legs were held in marked flexure contractures, equally on both sides. The arm-jerks were exaggerated, but the leg reflexes could not be elicited on account of the extreme contractures. The arms were œdematous from the elbow downward, and over the flexor and extensor surfaces of the elbows there were blisters, evidently trophic in origin. On the heel of the right foot, and the first metatarsal joint on the left, there was an area presenting a black appearance, and resembling local gangrene. There was no discharge or ulceration, and a similar lesion was cured previously, simply by the application of dry antiseptic dressings. There was incontinence of urine and feces.

The patient died of interstitial nephritis and cirrhosis of the liver a few days later. The brain and spinal cord were hardened in formalin, and sections from the various regions of the brain, pons, medulla, and cord were stained by the Weigert and Marchi methods and by hæmalum and carmine.

The pia of the paracentral lobule was slightly infiltrated with cells, and the bloodvessels were thickened. A slight perivascular round-cell infiltration was present in the cortex. In the frontal and occipital regions similar changes were found.

There was an area of softening in the pyramidal tract on one side of the pons, and the pia over the pons was the seat of a round-cell infiltration, and its vessels were thickened. The fibres of the pyramidal tract, near the area of softening, stained fairly well, except those which protruded upon the softened area. On the other side the fibres of the pyramidal tract were stained deeply. In the medulla on one side the pyramidal fibres were stained less deeply than on the other side. Here also the pia showed round-cell infiltration.

In the cervical region of the cord each crossed pyramidal and direct pyramidal tract stained by the Marchi method appeared much degenerated. This was also shown by the Weigert method.

A moderate, round-cell infiltration only was found in the pia of the cervical region of the cord. Amyloid bodies were present in large numbers in the posterior columns. In the lumbar region the crossed pyramidal tracts were slightly degenerated. The pia was infiltrated with round cells, but not intensely so, while within the cord a mild degree of perivascular infiltration was observed. The Marchi method showed a recent degeneration of the crossed pyramidal tracts. The microscopic findings were those of cerebrospinal syphilis. It should be noted here that the history of specific infection was denied.

CASE III.—C. B., aged fifty-seven years, a spinner by occupation, was admitted to the Philadelphia General Hospital, February 6, 1905. Her father is living and well. Her mother died of "heart failure." Two brothers are living and well. Two brothers died of phthisis, and one sister died suddenly.

She had borne eleven children, all of whom died in infancy. She never had any miscarriages. She suffered the ordinary diseases of childhood, and had always been temperate.

A year before her admission to the hospital there was a history of paralysis of the right arm and leg, from which she largely recovered. During the last week in January, 1905, three weeks prior to her admission, she suddenly lost power in the left arm and leg, without disturbance of consciousness. Since this time there had been no return of the power, in fact she gradually grew weaker.

When examined by myself March 20, 1905, she presented the following conditions: There was no facial palsy. The tongue was protruded straight. The grasps were both feeble, but the left more so. The left arm was spastic at the elbow and shoulder, while the right showed no tendency whatever to spasticity. The right leg was slightly spastic, and there were beginning contractures in the foot. Voluntary power in the right arm and leg was present only in a moderate degree. The left leg was held up across the body in a flexed position, the knee reaching the right side of the body. This contracture could be only partially overcome, and effort to do so was attended by pain. The arm-jerks on the right were unobtainable on account of the contractures present; on the left side they were exaggerated. The knee-jerk on the right side was exaggerated; on the left it could not be obtained, due to the contractures. The ankle-jerk was present on both sides, but no clonus could be developed either on the right or left. Babinski's phenomenon was present equally on both sides. There was no local atrophy discoverable anywhere. The urine and feces were passed incontinently. Sensation was intact. Examination of her lungs disclosed no marked abnormality.

The patient died April 4, 1905, of chronic nephritis, and at the autopsy a tumor of the brain was found involving the right frontal lobe anteriorly, and the median fissure was slightly displaced to the left.

The tumor measured about three centimetres in all directions. Sections from this tumor presented the characteristic appearance of a gumma. Sections from the several regions of the brain and from the medulla were stained by the Weigert hæmatoxylin method, and with hæmalum and acid fuchsin. The pia in the right and left paracentral regions was infiltrated with round cells, and some of the bloodvessels were thickened. In the cortex in these regions there was also some infiltration around the bloodvessels, and in the cortex below the diseased pia. A similar condition was found in the right and left occipital regions, but it was less marked. In the occipital region the cortex did not show anything abnormal. The pia covering the peduncles was infiltrated with round-cells, but the peduncles themselves were not pathological. There was a small area of softening in the left pyramidal tract of the pons, situated near the raphe, and the pia covering the pons was the seat of a round-cell infiltration. In the medulla the pyramid on the left side took the Weigert stain less intensely than on the right side, and the pia covering the medulla was moderately infiltrated with round cells. In the cervical, thoracic, and lumbar regions on the left the crossed pyramidal tract was degenerated. This was not very marked, and could be more readily seen by the naked eye than with the microscope. The cells in the lumbar and cervical regions were normal. The pia of the cord at the levels studied was thickened and infiltrated with round cells.

The pathological changes in this case were characteristic of cerebrospinal syphilis.

CASE IV.—S. D., aged fifty-two years, a widow, was admitted to the Nervous wards of the Philadelphia General Hospital, December 29, 1904. The family history was negative. She had suffered from all the ordinary diseases of childhood, and had had smallpox at the age of forty. Her health had always been good otherwise. She had one child, born at term, which died in twenty-four hours. She never had a miscarriage. For years she had been a steady, hard drinker.

A year ago she suddenly became unconscious, and upon awakening she was weak in the left arm and leg. From this she recovered largely, although there was some defect on that side. A few days before her admission she fell to the ground, was unconscious, and afterward was found to be weak on the right side.

When examined by myself, February 13, 1905, she was generally emaciated. She moved the right side of her face less readily than the left, although the forehead was wrinkled equally well on both sides. The tongue was protruded straight, and the speech was somewhat indistinct, but there was no aphasia. The pupils were equal and responded normally. Both arms were weak, and while the grasps were about equal, the movements of the left arm were more restricted than those of the right. There was distinct loss of power in both legs, in the left more than the right. She was unable to stand. Both arms and legs were distinctly spastic, and on the left side there were contractures at the shoulder, elbow, hip, and knee. These could be overcome with a moderate amount of force. The first two toes of the foot were hyperextended.

The reflexes were everywhere increased, and the elbow-jerk and biceps-jerk much exaggerated, and equally on both sides. The knee-jerks were large and spastic, and a rectus clonus could be developed on the right side. There was no ankle clonus, and the ankle-jerk was absent. Babinski's sign was present on the left side, but not on the right side, although at a previous examination by Dr. William G. Spiller he noted the presence of this reflex on both sides. There was much muscular soreness in the legs, not localized to the nerve trunks. A pinprick was felt distinctly all over the body. There was no local atrophy.

The patient died April 20, 1905. At the autopsy an excess of fluid escaped when the skull was opened, and the pia and arachnoid were œdematous, while the basilar vessels were thickened. On the right side there was a large area of softening involving the temporosphenoidal lobe. On the left side a small area of softening was observed in the occipital lobe. Unfortunately the brain was largely destroyed at the autopsy, and portions of the cortex from the occipital and temporal regions, and the pons and medulla and cord only, were saved for microscopic study. In the sections from the temporal region there existed a round-cell infiltration of the pia, and around the bloodvessels, extending into the cortex. The same condition was present in the sections from the occipital lobe. No degeneration was present in the cerebral peduncles, but the pia covering them was infiltrated with round cells, and the vessels at the base of the brain were much thickened. In the medulla, stained by the Weigert method, one of the anterior pyramids was slightly paler than the other one, while the other pyramid appeared to be normal. The pia and bloodvessels of the medulla presented the same condition as that found in the pia of the peduncles. In the cervical region there was some slight degeneration of both crossed pyramidal tracts, which was more marked on one side than on the other. The pia of the cord at this level was greatly infiltrated with round cells. Some perivascular round-cell infiltration was observed within the medulla and spinal cord. The nerve cells of the anterior horns of the cervical region were in good condition, although the thionin stain was not employed. In the lumbar region the crossed pyramidal tracts were slightly degenerated. The nerve cells of the anterior horns appeared to be normal. The pia at this level was also infiltrated with round cells. The lesion in this case was also that of cerebrospinal syphilis.

In two of these cases the paralysis of the opposite side followed the primary attack of hemiplegia in a year's time; in one, after three months; and in the other, six weeks. In all of the cases the characteristic microscopic appearances of cerebrospinal syphilis were seen.

After searching the literature rather carefully for cases of double hemiplegia, I was surprised to find very few references, and these rather unimportant, and adding but little to our stock of knowledge on this subject. It may be that such cases are not reported under the title of double hemiplegia, for they are not of very uncommon occurrence.

Jolly[15] cited an interesting case of hemiplegia of sudden onset followed later by paralysis in both legs, the left leg remaining palsied to the end, some motion still remaining on the right side. Sensory symptoms were present. At the autopsy a pure myelitic focus was found in the spinal cord in the upper thoracic region, giving rise to secondary degeneration.

A case is reported by Brown-Séquard of double hemiplegia, and one in the *Medical and Surgical Practice*,[23] September 18, 1869, the symptoms of which are not important enough to quote.

In one case, reported by Dawson,[8] a boy of four became suddenly paralyzed in the right arm, leg, and tongue. Later, as the paralysis was improved, a gradual rigidity and loss of control of the left arm came on. The brain was softened, especially the cerebellum. Further details of the postmortem examination were lacking.

Dunn[9] described a case in which there was right-sided hemiplegia, hemianæsthesia, aphasia, and right lateral hemianopsia, following an attack of unconsciousness. The symptoms improved greatly in ten days, and in three weeks the patient was as well as before the attack. Two years later, upon awakening in the morning, he was found to be paralyzed on the right side. There was partial hemiplegia, anæsthesia, and absolute blindness. The palsy improved to the normal in four weeks, but the sight remained greatly impaired.

Köbner[17] recorded a case, in a man, of right-sided paralysis of sudden onset. Later there was transient palsy of the left arm, and repeated attacks of paralysis on the right side, arm, leg, and face. At the autopsy there was found a syphilitic endarteritis, meningitis, and chronic encephalitis. There was a gumma in the pons, and secondary degeneration of the pyramids on the left side. The left lenticular nucleus was the site of encephalitis and a cyst. Areas of softening were also found in the pons.

CONCLUSIONS.—1. In brain syphilis small microscopic foci of softening may occur in the cortex, the result probably of cutting off of the blood supply, and giving rise to hemiplegic symptoms and mental disturbances.

2. Syphilitic inflammatory encephalitis, while described comparatively rarely, may occur, and is probably due to inflammation originating in the small vessels of the cortex by the syphilitic poison circulating in them.

3. The change in the bloodvessels is not always uniform. Sometimes the adventitia is more intensely involved and sometimes the intima.

4. Double hemiplegia, if not uncommon, is certainly not frequently described.

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ON AUTOSUGGESTION IN HYSTERIA APROPOS OF A CASE, WITH REMARKS ON SO-CALLED "HYSTERICAL INSANITIES."[1]

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The study of the innumerable physical and mental manifestations observed in individuals suffering from hysteria can apparently never be exhausted. The character, disposition, emotions, and general mental state of hystericals may undergo so many changes at various periods of their life that the history of each individual case would

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[1] Read before the College of Physicians, November 1, 1905.